of the sacrolumbar mass during activity, hence the painful cramp and its peculiar intermittent character. I have found no cases recorded of intermittent claudication of the lumbar region, nor have I observed a similar case. It seems to me, however, not improbable that other cases of this same type may occur and have been overlooked or misinterpreted and that some of the lumbar pains and cramps of advanced life may be dependent upon arteriosclerotic processes in the abdominal aorta or its lumbar branches.

ACUTE YELLOW ATROPHY OF THE LIVER IN CHILDREN, WITH REPORT OF A CASE.

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Acute yellow atrophy of the liver is a very rare disease. In fact, Osler[1] states in the third edition of his *Practice of Medicine* that he had never seen a case. Rolleston,[2] in twenty years had seen in the postmortem room or examined the liver of 7 cases. It occurs much less frequently in children than in adults, and for that reason the following case is considered worthy of report.

G. R., a female child, aged five years, was admitted to the children's ward of Lakeside Hospital, November 3, 1909, suffering from intense jaundice associated with stupor. Neither the family nor the personal history threw any light on the case. One week before admission she began to lose her appetite, and vomited several times a day, the last time she vomited being two days before coming to the hospital. During this time the child became more deeply jaundiced, the stools were clay colored, and the urine a deep yellow color. The parents stated that they had not noticed any blood in the vomitus or in the stools. During the last twenty-four hours she had been unconscious.

Physical examination showed a well-developed and quite well-nourished child, tossing about restlessly in bed. The skin and mucous membranes were deeply jaundiced, a small ecchymotic area was noticed in the left lower quadrant of the abdomen, and another on the outer side of the right arm just above the elbow. There were numerous small petechiæ over the lower extremities below the knees. The child was unconscious, but seemed hypersensitive when touched. The pupils were widely dilated and reacted sluggishly to light. The ears, nose, and throat were normal, but the tongue was heavily coated with a yellowish-white fur. Slight general glandular enlargement was noted. The temperature was 96.2° by rectum, the respirations were 30 to the minute, and somewhat shallow and irregular. Examination of the lungs revealed nothing abnormal. The pulse rate was 64; with the exception of some arrhythmia there was nothing of importance in the examination of the heart. The abdomen was somewhat distended, especially in the lower part, this being due to a full bladder. The spleen could be felt. The lower edge of the liver was not palpable; the upper border of hepatic dulness began at the fourth rib in the right mammary line, and at the sixth rib in the axillary line. The tendon reflexes were exaggerated, and there was some spasticity of the legs. The urine was dark yellow in color; specific gravity, 1017; alkaline, contained a faint trace of albumin, and bile was present in large quantity. The microscopic examination of the centrifugalized sediment showed many epithelial and hyaline casts and many leucin crystals. The leukocyte count was 22,400.

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[1] Practice of Medicine, 3d ed.

[2] Diseases of the Liver, Gall-bladder, and Bile-ducts, 1905.

The child vomited shortly after admission to the hospital, the vomitus being mixed with dark clotted blood. The stomach was then washed out and numerous blood clots came away in the washing. The child died two hours later.

AUTOPSY REPORT. *Anatomical Diagnosis.* Acute yellow atrophy of the liver, icterus, petechial hemorrhages (skin, omentum, mesentery, mediastinal tissues, epicardium, pleura, lung, Peyer's patches, mucous membrane of the stomach), cloudy swelling of heart, kidney, pancreas, and adrenals, stenosis of the cystic duct, thyroid hyperplasia.

Autopsy was performed nine hours after death. The body was that of a well-formed female child, 95 cm. in length. Postmortem rigidity was slight. There was marked postmortem staining over the dependent parts, with numerous petechial spots on the extremities below the knees. The skin everywhere was markedly icteric, the conjunctivæ bright yellow, the pupils widely dilated and equal. A dark brownish fluid was exuding from both nostrils; the ears were free from discharge. The superficial fat was moderate in amount and bright yellow in color.

The peritoneum was smooth. The fat everywhere, omental, mesenteric, and in appendices epiploicæ, showed numerous hemorrhages varying in size from a pin head to a dime. The hemorrhagic condition in the mesentery was very striking, and seemed to be equally distributed throughout, being most marked toward the attachment to the gut. The liver was small, pale in color, its lower border lying well above the margin of the ribs. The gall-bladder, ducts, and the appendix appeared normal. There was a small amount of free bile-stained fluid in the peritoneal cavity. There was nothing abnormal about the diaphragm.

Both pleural cavities were free from adhesions, and contained a small amount of bile-stained fluid. Beneath the visceral pleura, numerous small hemorrhagic points were noted; these were also quite numerous in the mediastinal fat and in the fat of the epicardium. The pericardium contained a small amount of bile-stained fluid; there were no adhesions, and no subpericardial hemorrhages. The heart weighed 70 grams. The valves were normal; the endocardium was bile stained, but there were no hemorrhages beneath it.

The right lung weighed 170 grams, crepitant and air containing throughout. The surface was smooth and mottled with small dark red areas. On section throughout the lung substance, varying in size from a pin point to 2 cm. in diameter, were numerous small hemorrhages; the bronchial mucosa was slightly congested. The left lung weighed 160 grams, and showed the same conditions as the right except that the hemorrhagic areas were more numerous. The bronchial lymph glands were not enlarged.

The colon was normal. The ileum showed nothing of note except a hemorrhagic Peyer's patch just above the ileocecal valve. The duodenum showed some congestion just above the papilla of the common bile duct. The bile ducts were normal with the exception of the cystic duct, which was occluded 2 cm. from its origin in the gall-bladder. The walls of the stomach were covered with a large amount of thick mucus, and there were a few small hemorrhages into the mucosa along the greater curvature.

The liver weighed 250 grams, was pale in color, very flabby, and showed some wrinkling of its capsule. It lacked the rigidity of a normal liver, and partially collapsed and bent under its own weight. On section, the lobular markings were indistinct, and showed bright yellow areas 1 to 2 cm. in diameter, mingled with reddish slightly depressed areas. The outlines of the lobules were entirely lost in the red and could only occasionally be discerned in the yellow areas. The gall-bladder contained a small amount of bright yellow mucus, and the walls were slightly congested. The pancreas was normal.

The right kidney weighed 80 grams. The capsule stripped readily, leaving a smooth surface, fetal lobulation was well marked. On section, the cortex was swollen, and showed marked cloudy swelling. The left weighed 75 grams and was similar to the right.

The thymus was normal. The thyroid was slightly enlarged. The trachea and larynx showed no submucous hemorrhages.

The brain weighed 1075 grams. The calvarium showed marked depressions, every convolution of the cerebrum being marked by a corresponding mould in the inner table of the skull. The dura mater showed definite bile staining. The section of the brain showed nothing unusual.

On microscopic examination, the liver showed extensive degeneration. In many sections no trace of liver cells could be seen, nothing being left but the vascular stroma. In other sections traces of liver cells still remained, but the cells were granular, vacuolated, and stained diffusely; an occasional cell could be seen which stained well, these having a granular vacuolated cytoplasm and a small darkly stained nucleus. There was no evidence anywhere of regeneration of liver cells. In the areas where the degeneration of liver tissue was most marked, the capillaries were distended with blood. Hemorrhages were quite numerous. The interlobular bile ducts showed everywhere a marked proliferation; this was most marked in the areas where the destruction of the liver cells was greatest. Here the lobules were completely surrounded by proliferated connective-tissue of Glisson's capsule in which were numerous newly formed bile ducts. These showed a tendency to spread down into the lobules in places, which was evidently an attempt at regeneration. Glisson's capsule showed a slight round-celled infiltration, and was everywhere thickened, in some places considerably so. The gall-bladder was normal; desquamated epithelial cells filled the lumen of the bile ducts. The heart showed nothing but a considerable grade of cloudy swelling. In the lungs a few small areas of focal hemorrhage were seen. There was nothing else abnormal. The thymus was normal. The thyroid showed a moderate grade of hyperplasia. A moderate degree of cloudy swelling was found in the adrenals. The brain was normal.

Cultures taken from the heart blood gave a pure growth of staphylococcus albus. Sections of the liver stained by the Levaditi method failed to show spirochetes.

In 1894 William Hunter was able to find 250 published cases of acute yellow atrophy of the liver. Four years later McPhedran[3] collected 29 additional cases. In 1903 Best,[4] in an exhaustive study, found about 500 cases reported in the literature. Of these, a small proportion occurred early in life. I have been able to find reports of 40 children with this disease; these including my own patient making a total of 41. In 1905 Rolleston was able to collect 22 cases in the first ten years of life.

One of the earliest cases recorded was that of Politzer,[5] who, in 1858, observed this condition in a newborn infant. On the fourth day after birth the child became ill with jaundice and bloody vomit, which became worse on the following day, and was accompanied by high fever and restlessness with a tendency to stupor. Later the left lobe of the liver showed a perceptible diminution in size. On the eighteenth day the child died, death being preceded by convulsions and bleeding from the umbilicus. The urine showed leucin and tyrosin cyrstals. The liver at autopsy was considerably diminished in size, the diminution chiefly affecting the left lobe. The bile passages were patent and the umbilical veins and arteries were normal. The microscopic examination of the liver showed a high degree of destruction of the liver cells. The same author, two years later reported a case in a girl baby four months old, who became suddenly ill with diarrhea, vomiting, and fever, the symptoms suggesting a gastro-intestinal catarrh. On the seventh day of the illness, icterus appeared, which became very intense. The stools were free from bile pigment. The liver was enlarged and painful on pressure. This increased liver dulness continued until the twelfth day. On the fourteenth day there was evident diminution in the size of the liver, blood was vomited and passed with the stools. Forty-eight hours later the child died from exhaustion, and by this time no liver dulness could be made out below the costal margin. An autopsy showed that the liver was small and flabby, and microscopically there was great destruction of liver cells, the latter containing a great deal of fat.

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[3] Sajous' Annual, 1899, iv, 393.

[4] Thesis, Univ. of Chicago, 1903.

[5] Jahrb. f. Kinderheilkunde, 1860.

The following cases have been reported by various authors: Loeschner,[6] boy, aged three years; Mettenheimer,[7] boy, aged four years; Widerhofer,[8] girl, aged one and three-fourths years; Hecker,[9] newborn girl; Senator,[10] girl, aged eight months; Brandenburg,[11] boy, aged eleven weeks; Hilton-Fagge,[12] boy, aged two and one-half years; Steiner,[13] boy, aged ten years; Monti,[14] boy, aged four years; Pleischl and Falwarczny,[15] boy, aged fourteen years; Mann,[16] boy, aged one and one-fourth years; Aufrecht,[17] newborn child, Dinkler,[18] girl, aged twelve years; Brunton and Tunnicliffe,[19] boy; aged three and one-half years; Foltanek,[20] boy, aged twelve years; Goodhart,[21] boy, aged two and one-half years; Bjelin,[22] child, aged thirteen years; Greves,[23] child, aged one and three-fourths years; Babes,[24] boy, aged six years; Lewitsky and Brodowsky,[25] child, aged fifteen years; Lanz,[26] boy, aged four years; Meder,[27] boy, aged fifteen years; Merkel,[28] boy, aged six years; Poddig,[29] boy, aged two and one-half years; Rehn and Perlo,[30] boy, aged two and one-half years; Rinsema,[31] girl, aged five years; Rosenheim,[32] girl, aged ten years; Heukelon,[33] child aged three months; Starck[34], boy, aged two and one-half years; West,[35] boy, aged six years; Schmidt,[36] boy, aged two and one-fourth years; Todt,[37] girl, aged three and one-yalf years; Carpenter,[38] girl, aged twenty-three months; Rogers,[39] boy, aged four years; Parkinson,[40] boy, aged four years; Wentworth,[41] boy, aged five years; Griffith,[42] boy, aged seven years; Drennan,[43] boy, aged eight years. The proportion of boys and girls afflicted is approximately two to one.

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[6] Oesterrichische Zeits. f. Kinderheilkunde, 1856. (Abstract, British and Foreign Medico Chirurg. Review, 1860, vol. xxv.

[7] Memorab. aus der Praxis., 1862, vol. vii.

[8] Jahrb. f. Kinderheilkunde, 1859, Band ii, p. 42.

[9] Monatssch. f. Geburtskunde, 1867.

[10] Jahrb. f. Kinderheilkunde, 1865.

[11] Festschrift f. e. Hagenbach-Burckhardt, Basel, Leipzig, 1897, S. 61.

[12] Trans. Path. Soc., London, 1869, xx, 212.

[13] Jahrb. f. Kinderheilkunde, 1871, S. 428.

[14] Archiv f. Kinderheilkunde, Band vii, S. 346.

[15] Zeits. der K. K. Gesell. der arzte zu Wien, 1858.

[16] Amer. Jour. Obstet., 1875, viii, 539.

[17] Zentralbl. f. innere Med., 1896, No. 2.

[18] Inaug. Dissert. Halle, 1887.

[19] St. Barthol. Hosp. Rep., 1896, xxxii, 425.

[20] Quoted by Merkel.

[21] Trans. Path. Soc., London, vol. xxxiii, p. 82.

[22] Quoted by Merkel.

[23] Brit. Med. Jour., 1884, i, 766.

[24] Virchow's Archiv, 1894, No. 136.

[25] Quoted by Merkel.

[26] Wien. klin. Woch., 1896.

[27] Ziegler's Beiträge z. Path. Anat., 1895, Band xvii, S. 143.

[28] Münch. med. Woch., January 30, 1901.

[29] Inaug. Dissert. Königsberg, 1892.

[30] Berl. klin. Woch., 1875, S. 644.

[31] Quoted by Merkel.

[32] Zeits. f. klin. Med., 1889, Band xv.

The etiology of acute yellow atrophy of the liver is quite unknown, but it is probably not due to a specific poison, for we find that numerous forms of intoxication, particularly chloroform, phosphorus, syphilis, alcohol, puerperal eclampsia, may lead to a condition closely resembling this disease. I have seen one case of chloroform poisoning in which the symptoms and the course of the disease were indistinguishable from acute yellow atrophy of the liver, and leucin and tyrosin crystals were found in the urine. Inasmuch as there is such a marked resemblance between this disease and phosphorus poisoning, some writers have thought that all cases were due to poisoning with phosphorus, but that is not true, as recent pathological studies have served to differentiate between the two conditions. Wells[44] has summarized the chief differences between phosphorus poisoning and acute yellow atrophy as follows: "Phosphorus produces a general injurious effect upon all the organs of the body, the liver merely showing the most marked anatomical changes, which at first consist of fatty metamorphosis of the liver, due to a migration of the body fat from the fat deposits into the injured cells, subsequently the liver cells disintegrate, the cytoplasm being affected before the nucleus, and the liver may become smaller than normal, although it is usually enlarged because of the fat deposition. Typical acute yellow atrophy is characterized by an early necrosis of a large proportion of the liver cells, the nucleus becoming unstainable while the cytoplasm is still little altered in appearance, and fatty changes play a subordinate role or are absent. As Auschutz says, "The poison seems to strike at the life of the cell, its nucleus, while phosphorus attacks the cytoplasm. Furthermore, the poison of yellow atrophy seems to be very specific, for it attacks the other organs of the body almost not at all, and within the liver it affects only the hepatic cells proper, while the bile duct epithelium and stroma cells are so little injured that they are able to proliferate greatly, this proliferation being a prominent feature." Wells further considers that the atrophy is due to an autolysis of necrotic liver cells by their own enzymes, and that in the most typical cases of acute yellow atrophy we have to do with a poison having a very specific effect on the liver cells, which destroys their synthetic activities without injuring their intracellular proteolytic enzymes, and consequently autolysis occurs. The fact that so many cases are preceded by symptoms of gastro-intestinal disturbance would lead us to suppose that this poison was of intestinal origin. Quincke considered that possibly regurgitation of pancreatic juice up the bile ducts might be responsible for the degenerative condition in the liver, but this is not true, for the bile pressure is greater than that of the pancreatic juice, and the bile ducts and peripheral portions of the lobules are always least affected. Klebs, Eppinger, and others have ascribed a bacterial origin to this disease, basing their conclusions on the finding of bacteria in the bile ducts and liver detritus in fatal cases. The bacteria in these cases were probably secondary invaders. In a case reported by Aufrecht, the disease in a newborn infant was associated with a sclerema neonatorum.

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[33] Nederl. Tijdschr. v. Geneesk., 1888.

[34] Jahrb. f. Heilkunde, 1898.

[35] Trans. Path. Soc., London, 1880, xxxi, 116.

[36] Inaug. Dissert. Kiel, 1897.

[37] Akute gelbe Leberatrophie im kindlichen Lebensalter. Berlin, 1903, p. 32.

[38] Reports of Soc. for Study of Disease in Children, London, v, 133, 134.

[39] Reports of Soc. for Diseases in Children, 1906, vi, 294.

[40] Report of Soc. for Study of Disease in Children, London, 1906, vi, 229.

[41] Archives of Pediatrics, February, 1906, p. 81.

[42] Archives of Pediatrics, February, 1906, p. 88.

[43] Jour. of Path. and Bact., July, 1911, p. 141.

[44] Chemical Pathology, 1907.

Acute yellow atrophy may occur in chronic drunkards, the condition supervening on existing cirrhosis. It may also occur in secondary syphilis. In a case reported by Merkel, though the child showed no signs of syphilis, the father had a syphilitic spastic paralysis of the lower extremities, and the mother had had repeated abortions. The disease is commonest between the ages of twenty and thirty in adults, females being more often attacked than males, in the proportion of 2 to 1, in contrast to the condition we see in children, where males are more frequently affected.

The majority of cases run their course in two weeks. Occasional cases occur in which the disease is spread over many weeks or months. In general we can divide the symptoms into two stages. The first, which lasts five or six days or sometimes much longer, is characterized by malaise, often muscular pains, nausea, vomiting and occasionally diarrhea, at other times constipation. The jaundice appears during the latter part of this stage. Often the jaundice is the first sign, increasing steadily in its intensity. Cases of acute yellow atrophy have been described in which no jaundice was present.

The second stage is marked by the pronounced grave change in the patient's condition, and especially by the appearance of nervous symptoms, severe headache, intolerance of light, restlessness, delirium, twitching of the muscles, convulsions, coma. In this stage transient paralyses sometimes occur, so that, excluding the jaundice, the clinical manifestations may closely resemble meningitis.

With the onset of the second stage the vomiting becomes more severe and the vomitus often contains blood. The tongue is dry, brown, and tremulous, and the teeth covered with sordes. The pulse is rapid and of low tension. The respirations are often quite irregular. The temperature is often elevated; in other cases it is depressed, rising just before death. Often petechiæ and hemorrhages are found beneath the skin, and blood may be passed in the stools and urine. Epistaxis occasionally occurs. The liver at first may be increased in size, but, as a rule, the liver dulness is much diminished before death, this diminution being partly due to the change in size of the organ and partly to its flabbiness, allowing it to fall away from the abdominal wall, the coils of intestine taking its place. The spleen is sometimes enlarged. The urine contains a great deal of bile and, as a rule, albumin and casts. It is remarkable that glycosuria is not observed. The urea is diminished, but the percentage of nitrogen present as ammonia is increased. Leucin and tyrosin are nearly always present, sometimes one being present in the urine without the other. There is nothing distinctive about the blood. Leukocytosis is nearly always present, and in a few cases colon bacilli have been found by blood cultures. The coagulability of the blood is decreased.

Morbid Anatomy. The liver is always greatly diminished in size, often weighing only one-half to one-third of the normal weight. The liver is more or less universally affected, but often the left lobe shows the most marked atrophy, as in the first case reported by Politzer. In the chronic cases where the disease has existed for months the surface of the liver, instead of being smooth, is nodular, due to a compensatory hyperplasia of the liver cells, comparable to the adenomata seen in cirrhosis. The capsule is wrinkled and loose, and small hemorrhages may be seen beneath it. The liver is flabby, so that it bends readily under its own weight. Its surface is greenish yellow in color, with here and there reddish areas. It cuts with resistance. The cut surface is a bright lemon-yellow color, with numerous patches of darker brownish red, the latter predominating in the left lobe, where the greatest atrophy is seen. The yellow areas are soft and slightly swollen, the reddish areas are tough and of a leathery consistency. Histologically the change in the yellow areas are seen to be more recent, the parenchymatous cells showing extensive fatty changes, the fat droplets being within the nucleus, as well as within the cytoplasm. In the red areas, absorption of the fatty material has taken place, and we see mainly a network of capillaries, within the meshes of which are degenerated liver cells and the detritus of broken-down liver cells, masses of chromatin, and blood. When death has been delayed, evidence of regeneration can be seen in the form of patches of newly-formed liver cells, with deeply staining nuclei, some of them exhibiting mitoses. Pseudo-bile capillaries may also be present in considerable number. Crystals of leucin and tyrosin may be seen in fresh sections, and the amount of fat which can be extracted from the liver is about 5 per cent. above the normal. The gall-bladder contains bile mixed with a great deal of mucus, and the bile ducts are normal.

The kidneys and heart show evidences of cloudy swelling, and the spleen is softened and enlarged.

The gastro-intestinal tract usually shows evidences of catarrhal inflammation. There is often some ascites.

Petechial hemorrhages may be found beneath the pleura and pericardium, and in the intestinal mucosa, and sometimes extensive cerebral and meningeal hemorrhages occur.

The diagnosis is usually simple from the intense jaundice, the nervous symptoms, the atrophy of the liver and the finding of leucin and tyrosin crystals in the urine. The latter are sometimes seen in other conditions of the liver, where there are marked degenerative changes. Sometimes these cases are confused with cases of phosphorus poisoning, but a careful history will serve to distinguish the latter. Hilton-Fagge has recorded a case in a boy, aged two and one-half years, where the symptoms were incorrectly thought to be due to belladonna poisoning.

Acute yellow atrophy of the liver is almost uniformly fatal. A few cases of undoubted recovery have been recorded. In 1897 Wickham Legg gave a list of 28 reported cases of recovery.

There is no treatment of much avail. Free purgation, intravenous or subcutaneous transfusion with saline solution, and intestinal antisepsis have been recommended.

TREATMENT OF NOCTURNAL ENURESIS IN CHILDREN.

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Nocturnal incontinence of urine in children is one of the common and one of the most troublesome conditions which the physician is called upon to treat. It is perhaps for this reason that it is so commonly neglected both by the profession and by the laity. The physician after one or two therapeutic ventures dismisses the case with the suggestion that the adenoids should be removed, or, if it is a boy, that he be circumcised, and then if these suggestions are carried out and a cure does not result the family of the child become resigned to what they regard as inevitable.

If we exclude at the outset those cases in which there is evi-